Arrhythmias and particulate matter

See Articles page e58

There is strong epidemiological evidence that inhalation of combustion-derived soot triggers life-threatening cardiovascular events. Indeed, the 2015 Global Burden of Disease estimated that 1 521 000 (35%) of the total of 4 241 000 deaths attributable to ambient particulate matter (PM; all causes) were from ischaemic heart disease.[1] But policy makers continue to regard these mortality figures with scepticism, and delay enacting the urgent interventions that will significantly reduce population exposure to air pollution. For example in the UK, there is no national plan to replace the present highly polluting fleet of diesel cars, vans, and taxis. Potential reasons for this scepticism include difficulty in communicating to non-specialists the methods used to attribute deaths to air pollution,[2] and the absence of experimental data directly linking inhalation of PM to cardiovascular disturbances known to cause fatal events. The study by Franco Folino and colleagues, addresses this latter issue by linking air pollution data from the Vento region of Italy with ventricular arrhythmia data stored by implanted defibrillators. Folino and colleagues report a significant association between episodes of ventricular tachycardia and ventricular fibrillation and PM of less than 2·5 μm in aerodynamic diameter ($PM_{2·5}$),[3] a result that both strengthens the case for a causal association between PM and cardiovascular deaths, and suggests further innovative studies. First, by adding personal carbonaceous PM monitoring to this group of high-risk patients (figure), it should be possible to determine the independent association between locally generated traffic PM and arrhythmias. Second, contribution of fossil fuel and wood smoke to background PM in the Vento region offers a way of assessing the effect of different types of PM. Indeed, in-vitro toxicity studies suggest that not all particles are equal. For example, wood smoke PM, compared with diesel PM, induces higher levels of oxidative damage to human umbilical endothelial cells and stimulates increased monocyte adhesion.[4] Normally, ambient black carbon from fossil-fuel combustion is measured by an aethalometer using infrared light absorption. However, some of the larger aethalometers also assess ultra-violet light absorption, a spectrum that detects aromatic organic compounds found in wood smoke and other biomass sources. Thus, light absorption from both wavelengths could be used to identify the independent effect of background wood smoke PM on arrhythmias—a result that would be highly relevant to exposures in low-income countries, where wood burning is a major source of personal exposure to carbonaceous PM.[5]

What should clinicians tell high-risk cardiovascular patients about air pollution? To date, management guidelines provide little or no guidance. A recent workshop hosted by the European Respiratory Society addressed this question and identified two major areas that should be considered by future guidelines.[6] First, patients should understand how acute episodes of poor air quality will affect their own health, and the actions they need to take to protect themselves.

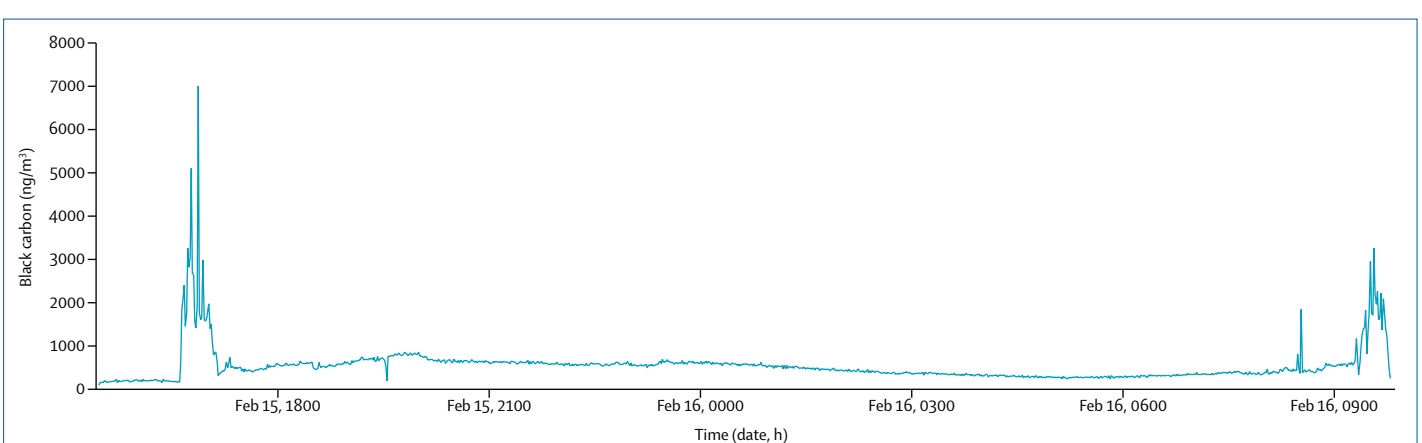

***Figure*: Data from a portable aethalometer carried for 24 h by an adult working in London (UK)**
Peaks of black carbon (BC; ng/m³) exposure occur during commuting, with relatively low exposures at the home address (2100 h to 0600 h).

Second, health-care providers need the tools to advise patients about air pollution and how to change their treatment plans or activities on high pollution days, and how to reduce long-term exposure.[6] In some cities, patients are able to sign up to text message services that warn about pollution and provide advice about how to reduce short-term risk. For example, London's airText's advice to adults with heart problems on very high pollution days is to "avoid strenuous physical activity".[7] It remains unclear, however, whether this, albeit sensible, advice reduces the daily dose of particles inhaled into the lungs. Use of a facemask on high pollution days is another option that patients are increasingly asking clinicians about. Facemasks are socially acceptable in countries such as China, and are widely used by urban cyclists in many other countries. Preliminary evidence for the efficacy of facemasks was provided by a randomised crossover trial[8] of patients with coronary heart disease who walked a predefined route in central Beijing, China, with and without a facemask. The study found that the facemask was well tolerated, reduced maximal ST segment depression, and increased heart rate variability.[8] Avoidance of pollution hotspots from local road traffic is a possible way of reducing long-term exposure. The UK charity Global Action Plan combined short-term and long-term advice in its patient information pack.[9] The pack includes a map of local high-pollution streets, and recommends regular use of less polluted routes (using the map), travel outside the rush hour, and regular checking of the daily air pollution forecast. In summary, we urgently need more evidence to identify the most effective personal exposure reduction strategies. In developing this evidence, clinicians should not forget to continue to challenge governments to implement national policies that significantly reduce air pollution.

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Jonathan Grigg is part-funded by the NIHR North Thames Collaboration of Leadership in Applied Health Research and Care.

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